M E M O R A N D U M

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The external iliac artery is a common site for atheromatous plaque formation especially at the level of the inguinal ligament. This is also the approximate level at which the iliac artery becomes the femoral artery (see attached). The location of alledged obstruction and the plaintiff's age of 60 years dismisses a possible claim of Buerger's Disease.

Regarding the possibility of tobacco associated atherosclerosis, there are claims (i.e. Caro, et al., Lancet, July 4, 1987, page 11-13) that cigarette smoking contributes to atherosclerosis development by increasing arterial wall stiffness and reducing blood flow pulsatility. This same paper concedes that the mechanism by which smoking causes these effects is unknown.

Some claim that cigarette smoking promotes atherogenesis by the actions of the constituents of smoke such as nicotine. Nicotine's vaso-constrictive actions have been implicated in exacerbating atherosclerotic formation (Whittemore, et al. 1987, attached). Some claim that ETS provides physiological levels of nicotine for vasoconstriction of peripheral blood vessels, most of the same studies indicate that these levels are not sufficient to cause changes in heart rate or blood pressure (Ohkubo, 1982, attached). The adverse effects of carbon monoxide and its role in precipitating atherosclerosis by endothelial damage or by increasing the viscosity of blood (Weir and Fabiano, 1982) are well known. However, other studies which consider the many other possible sources of carbon monoxide indicate that the carbon monoxide of passive smoking poses negligible health hazard (Cole, 1981). Cigarette smoking has also been implicated in causing a slight to moderate rise is total serum cholesterol, specifically increasing LDL and decreasing HDL (Dousett, et al., 1986).

Important to remember is that most all of these studies and claims of cigarette smoking or ETS contribution to atherosclerosis are pertinent to coronary vessel disease and few if any studies on peripheral vascular diseases have been conducted (Schievelbein, et al., 1984, attached). Some other contributing

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factors which have been implicated in coronary vascular disease and extrapolated to peripheral atherosclerosis include hypercholesterolemia as previously mentioned, diabetes, hyperlipoproteinemia, obesity, age, stress, and hypertension. Sharon Brock's treatise, Cardiovascular Disease, Foundations and Preparations, 1987 is a good background for atherosclerosis.

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Attachments

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